

causing local pressure on, or laceration of, the brain should be elevated. In all cases due regard should be paid to the treatment of general shock.

4. Compound fractures of the skull, with penetrating wounds of the brain, should be cleansed early. Gross foreign material, including bone, should be removed, and drainage provided.

5. Reduce intracranial hypertension by free catharsis, and, if necessary, by spinal puncture.

6. The diagnosis of active hæmorrhage from the middle meningeal artery is a positive indication for early operation.

7. Depression of a portion of the inner table of the skull without clinical evidence of local

pressure on the brain is not an absolute indication for operation.

8. Clinical signs of general cerebral hypertension do not constitute an absolute indication for craniotomy in the absence of definite signs of removable local pressure.

In short, I would urge conservative, symptomatic treatment.

Observation of ex-soldiers has taught me that of those who received serious wounds of the head comparatively few have developed epilepsy; and of those soldiers who have developed epilepsy comparatively few are men who were seriously wounded about the head.

MENINGITIS SEROSA CIRCUMSCRIPTA

WITH REPORT OF A CASE

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THE surgeon who is called upon to deal with disorders of locomotion must develop at least a nodding acquaintance with neurology. Cases of spastic paraplegia are frequently seen by him, the majority of them occurring in children. Most of these are congenital, and are classified eponymously as "Little's Disease." In young adults disseminated sclerosis frequently presents the picture of spastic paraplegia. In later life a spastic ataxia may be manifested at an early stage of pernicious anæmia. Somewhat less common, but capable as a rule of fairly easy recognition, is the group of lesions known collectively as Friedreich's ataxia. As a souvenir of an attack of transverse myelitis, the condition may be seen; as a sequel to an attack of epidemic cerebrospinal meningitis it is somewhat more rare. Such a case was recently under the care of the writers, and stimulated a search in the literature which resulted in identifying the picture presented with a group described under the name of "Meningitis serosa circumscripta." It may be well to present the history of this patient before discussing the disease.

H. C., male, aged 16, a student, came under the care of Dr. Dawson of Piapot, Sask., on January 9, 1927. The case history, as received from Maple Creek Hospital, is as follows:—

"Sudden onset; pain in head, extreme restlessness, delirium, marked albumen in urine on January 9th; immediately transferred to hospital. On January 11th complained of stiffness and pain in neck and back, retraction. Kernig's sign was present. Lumbar puncture was done, but no fluid could be obtained.

"Transferred to a separate room and treated as an isolated case with special nurses. He was given antimeningococcus serum 20 c.c. intravenously on January 11th. He ran a course of extreme restlessness and delirium for several days. Serum was given again (20 c.c.) on January 13th. After January 16th the symptoms were less acute and the patient was rational at times; there was general improvement until January 23rd, when he seemed to suffer a relapse with recurrence of all symptoms, but more severe than previously. Marked twitching (general), irrational, very drowsy and almost comatose, with very restless intervals until February 3rd, when decided improvement commenced. During all this time the patient was very helpless and from February 3rd on was depressed and very irritable, but gradually improved. He went home on March 9th on a stretcher, still unable to get up. (F. B. Dawson).

"The temperature on admission was 103.8°;

pulse, 88; respirations, 24. The temperature gradually settled to about 100, but rose again in conjunction with the relapse, finally reaching normal about the end of the first week in February and remaining so until his discharge. Albuminuria was not present after January 11th. He was discharged with the diagnosis of cerebro-spinal meningitis, a diagnosis justified by the clinical course of the case."

On October 17, 1927, the patient was referred to us by Dr. M. D. Mitchell, of Piapot, Sask., on account of weakness in the lower limbs.

The history obtained from his mother was that he had remained in bed for four months altogether, and was then able to go about. About two months before he was examined he had noticed weakness in the legs. This had become gradually worse. A curious statement was that on one occasion he had jumped out of bed to put out his light and felt as if something warm passed down both legs. He was able to move his lower limbs quite well, but by the morning they were again weak.

On examination the patient was found to be a well-formed lad of sixteen. The central nervous system showed a bilateral Babinski, and the Chaddock and Gordon reflexes to be present. On the right side ankle clonus was present; on the left side the ankle jerk was very brisk. On both sides the knee jerk was very brisk. There was no patellar clonus. The cremasteric reflexes were sluggish. The abdominal reflexes were faint on the left side, absent on the right side. Examination of sensation showed a patchy anæsthesia and analgesia, and patchy loss of thermal sensation, the thermal anæsthesia not corresponding to the cutaneous anæsthesia. Voluntary movement was present in all muscles of the lower limbs, but was weak and spastic in character. There was marked ataxia, amounting to almost complete loss of balance. Even with the eyes open the patient could progress only by staggering and holding on to one object after another.

On October 20th, a lumbar puncture was done but no fluid was obtained. The presence of root pains while the needle was in the spinal canal suggested that this was a *bona fide* "dry tap." The injection of lipiodol was considered, but, influenced, probably unduly, by unfavour-

able reports of this procedure, we rejected the measure.

The sensory dissociation suggested that the condition might be disseminated sclerosis, but the history of a preceding attack of meningitis, the lack of success in obtaining fluid by lumbar puncture, and the history of transient restoration of power, inclined us to the diagnosis of meningeal adhesions. As the outlook apart from operation was as gloomy as possible, it was decided to do an exploratory laminectomy, the site of exposure centring over the 11th thoracic vertebra.

On October 22nd, the laminae of the 10th, 11th and 12th thoracic and the 1st lumbar vertebrae were removed. The dura mater appeared to be normal. It was incised longitudinally and retracted. It was not adherent to the pia-arachnoid. The picture then presented was an exceedingly interesting one. Fluid was seen in the subarachnoid space down to a level corresponding to the upper part of the 12th thoracic body. This fluid pulsated. At this level there was a whitish mark where the pia-arachnoid was bound down to the back of the cord, and below this level there was a very small amount of fluid, not enough to show pulsation. The arachnoid was opened, the adhesion was dissected off the back of the cord and found to extend around it as far as could be reached. No attempt was made to follow the leptomeninges along the nerve roots. Removal of the adherent membranes on the back of the cord left a small rough area which showed tiny bleeding points. The dura was allowed to fall back into position but was not sutured. The soft parts were sutured in the usual way.

Two days afterwards, *i.e.*, October 24th, the plantar reflex was normal on both sides and ankle clonus had disappeared. By October 25th all pathological reflexes had disappeared, though sensation remained unchanged. By October 27th sensation had returned as low as the ankle, and by November 4th no abnormality of sensation could be detected. The thermal sensibility was the slowest in recovering. On November 5th, patient was permitted to walk and this he did without any noticeable unsteadiness. He was kept in hospital until November 18th, showing steadily increasing ease of locomotion. When discharged on this

date he could walk quickly, turn round smartly, and stand with his eyes closed if his feet were eight to ten inches apart, but he showed slight unsteadiness if both feet were close together. A recent letter from his doctor states that he is having pain in the thoracic region. The doctor is inclined to attribute this to unduly hard work in the harvest field.

COMMENT

There are several interesting points in the case history: (1) the patchy character of the anaesthesia; (2) the apparent sensory dissociation; (3) the presence of a localized ring of adhesions, binding the arachnoid to the spinal cord and shutting off the cerebro-spinal fluid from the cauda equina; (4) the completeness and the speed of recovery after removal of the adherent ring. This was so dramatic as to be almost startling.

During the last few years a recrudescence of interest in the subject of meningeal adhesions has made its appearance. A number of cases have been presented before the Royal Society of Medicine, and comprehensive articles have been written by Stookey and by Armour. Our personal experience is limited to this one case, and the résumé of the condition which follows is derived from the writings of others.

Until recently these cases have not been sharply distinguished from cases of so-called "cysts of the arachnoid," nor is there yet a name for the condition upon which all are agreed. Stookey speaks of "Adhesive spinal arachnoiditis." In England the term "Circumscribed serous meningitis" is used.

PATHOLOGY

Adhesions are present, tying the membranes down to the surface of the spinal marrow. These adhesions may be single or multiple, circumscribed or extensive. From Armour's paper it would appear that the dura may also be adherent. In the present case it was not.

Both sections of the United States (far west and east of the Rockies), as well as Canada, show declines in the death rates from three diseases which are of paramount public health interest, namely, typhoid fever, tuberculosis and conditions incidental to pregnancy and childbirth. All indications are that new minimum death rates for all time will be recorded for these diseases.

ETIOLOGY

In regard to etiology, this is by no means clear. Horsley thought of the gonococcus as the infective agent. Others have recorded a preceding attack of typhoid, influenza, encephalitis, or meningitis. Tuberculosis has not been recorded. The Wassermann reaction has been negative in all the cases reported. Armour, citing Mauss and Kruger, makes a plea for trauma as an important factor. There is no evidence of coincident disease of the cord.

SYMPTOMATOLOGY

Cases are said to be most frequent in the fifth and sixth decades. The majority of cases have been of long standing and slow development. They have simulated very closely an extra-dural tumour of the spinal cord. Stookey suggests that differentiation may be made by lumbar puncture, which is likely to show a block in all cases, the block being either partial or complete. Xanthochromia he states is never present and the protein content of the cerebro-spinal fluid is not increased to a marked degree. The presence of xanthochromia and increased protein content is bound up with venous stasis, and this is absent in the case of spinal adhesions. Other observers note the presence of xanthochromia and increased protein content. In the present case no cerebro-spinal fluid was obtained.

The nervous signs are characterized by the diffuseness of the sensory changes, and their variability on examination at different times. The motor disturbances are a prominent feature, with exaggeration of the deep reflexes, and, in the majority of cases, the presence of pathological reflexes.

TREATMENT

The treatment is exclusively surgical. If it is possible to remove the adhesions completely, the result may be striking, as in the present instance; where the process is widespread and diffuse, the prospects are much less rosy.

Last year the weight of the terrible death toll from typhoid in Montreal and environs was reflected in a death rate of 35.0 per 100,000 among Canadian policyholders, up to the end of August. This year, the figure for the corresponding period is only 5.9—*Rep. Metropolitan Life Insurance Company*.